nervous systems in the occurrence of myocardial damage following aneurysmal subarachnoid hemorrhage (SAH), we measured serum levels of MB-CK (MB), myosin light chain (Mn) and troponin T (Tn), and plasma concentrations of noradrenaline (NA), adrenaline (ADR) and MHPG as an indicator of NA activity of the brain in 720 SAH patients who admitted within 24 hours of the onset. Eighty patients had elevated levels of MB, Mn or Tn that indicated myocardial damage (group A) and were compared to 640 patients without myocardial damage (group B). Myocardial damage in group A was related to the initial level of neurological severity, as assessed by WFNS grade, but was not related to the amount of subarachnoid blood visualized on the computed tomogram. There was no significant difference between two groups in blood pressure. Heart rate was significantly higher in group A than in group B (93 ± 29 vs 85 ± 20 /min; $p < 0.01$). Plasma concentrations of NA, ADR and MHPG were significantly higher in group A than in group B (2316 ± 4778 vs 828 ± 736 pg/ml; $p < 0.01$, 752 ± 1129 vs 362 ± 512 pg/ml; $p < 0.01$, and 23 ± 26 vs 9 ± 6 ng/ml; $p < 0.01$, respectively). In all patients, serum MB level had positive correlation with plasma NA, ADR and MHPG ($r = 0.48$, $r = 0.50$ and $r = 0.53$, respectively), and Tn level also had positive correlation with plasma NA, ADR and MHPG ($r = 0.75$, $r = 0.74$ and $r = 0.72$, respectively).

These results suggest that noradrenergic activities in the central and peripheral nervous systems play an important role in the occurrence of myocardial damage associated with SAH.

916-121 Three-Dimensional Volume Measurements of Plaques in the Arteria Carotis and the Arteria Femoralis

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Non invasive plaque quantification and assesment of progression are main goals in ultrasound diagnostics.

In 34 patients 114 ultrasound investigations were performed by three different methods, using a 5 MHz linear array (Vingmed Masters Series). The probe was combined with a three dimensional (3D) reconstruction facility (TOMTEC). The documentation was realized by ECG- and breath-triggered storing of the images in a transversal view. For optimal visualisation an angle correction up ± 30 degrees was possible. The plaque volume was calculated based on serial transversal sections by manual tracing of the plaque outlines. Each plaque was measured in one chosen mode by two experienced sonographers and once in another mode by the first sonographer. To validate the method we examined an in vitro plaque model.

Results: We measured 39 subsignificant plaques (< 40% lumen reduction) in 29 carotid and 9 femoral arteries. A mean plaque volume of 77.5 cmm in examination I, 83.5 cmm in examination II, and 78.3 cmm in examination III was calculated. The interobservervariability ranged from 0.07 to 20.6 (m 6.5)%, the intraobservervariability from 0.02 to 18.7 (m 8.1)%. The in vitro model with a defined volume of 1000 cmm was calculated between 931 and 1106 (m 1044) cmm with a mean interobserservariability of 6.37%. Additionally three dimensional reconstructions have been performed.

In conclusion three dimensional plaque volume measurement is a promising method for early detection of plaque progression in consecutive scans. Additional 3D reconstructions render a better conception of the plaque extention.

916-122 Myocardial Involvement in Takayasu Arteritis

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Congestive heart failure (CHF) is not an uncommon presentation in Takayasu arteritis (TA). To understand the pathogenesis of CHF in this disease we carried out endomyocardial biopsy (EMB) in 85 patients (males 29, females 56, age 2 to 42 yrs) with angiographically proven TA. The EMB was done form RV using cordis bioptome and 3 to 5 pieces were taken in 10% formalin for histological examination.

Myocardial involvement was present in 65 (76.3%) and included focal/diffuse myocarditis in 26 (30.5%), myofiber hypertrophy in 39 (45.8%) and endocardial thickening in 9 (10.6%). 39/65 (60%) with histological myocardial involvement had CHF. In 10 of these there was no associated hypertension or valvular involvement to account for CHF. Distribution of myocardial involvement did not show any predilection for the various types of TA as defined by Ueno et al. Immunosuppressive therapy resulted in regression of myocarditis on repeat EMB and improvement of CHF.

Conclusion: Myocardial involvement appears to be common in TA and EMB should be done in all patients with CHF to detect underlying myocarditis. In view of its therapeutic importance it should be incorporated in classification of TA.

917 Vascular Tone: Endothelin, Angiotensin II, and Nitric Oxide

Monday, March 25, 1996, Noon–2:00 p.m.
Orange County Convention Center, Hall E
Presentation Hour: Noon–1:00 p.m.

917-116 Pulmonary Clearance of Circulating Endothelin-1 in Dogs in Vivo: Exclusive Role of ET_B Receptors

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The pulmonary circulation plays an important role in the removal of circulating endothelin-1 (ET-1). Plasma ET-1 levels are increased in heart failure, pulmonary hypertension and congenital heart diseases and may partly result from reduced pulmonary clearance of this vasoactive peptide. We studied the role of the ET_A and ET_B receptors in the pulmonary clearance of [125]-I-ET-1 in anesthetized dogs using the multiple indicator dilution technique. Experiments were carried out before and 5 minutes after intrapulmonary injection of specific antagonists for the ET_A (BQ123) and ET_B receptors (BQ788) *(approximately 1 μmol per injection)*.

	Control	BQ123	Control	BQ788
	N = 5		N = 6	
Flow (ml/s)	42 ± 10	42 ± 9	56 ± 26	51 ± 24
HR (beats/min)	168 ± 20	169 ± 23	166 ± 7	167 ± 5
MAP (mmHg)	149 ± 23	143 ± 17	141 ± 13	140 ± 13
MPAP (mmHg)	14 ± 2.7	14 ± 2	20 ± 2	20 ± 3
Clearance (%)	36 ± 4.2	34 ± 6.4	33 ± 7	0 ± 2*
K_{seq} (s^{-1})	0.050 ± 0.009	0.047 ± 0.014	0.052 ± 0.019	–

*$p < 0.01$ vs control; MAP = mean arterial pressure; MPAP = mean pulmonary artery pressure; K_{seq} = pulmonary sequestration rate constant for ET-1.

ET_A and ET_B antagonists had no effect on systemic and pulmonary hemodynamics. Pulmonary clearance was unaffected by BQ123 but was completely abolished by BQ788 (from 33% to 0%), indicating that ET-1 is cleared exclusively by ET_B receptors.

Conclusions: The ET_B receptor is exclusively responsible for pulmonary ET-1 clearance in vivo. Alterations or downregulation of this receptor may contribute to the increase in circulating ET-1 levels found in certain pathologic conditions. These findings may also explain the observed rebound phenomenon after withdrawal of non-specific ET_A and ET_B antagonists.

917-117 Captopril Reduces Endothelin-1 Release From Human Umbilical Vein Endothelial Cells by Reduction of Angiotensin II Synthesis

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Angiotensin converting enzyme (ACE) inhibitors reduce AT II synthesis and increase bradykinin (BK) levels. Angiotensin II (AT II) stimulates the synthesis of endothelin-1 (ET-1). Elevated BK levels augment the release of nitric oxide that is thought to be a major functional antagonist of ET-1 and decreases its synthesis. This study was conducted to determine whether the reduction of AT II induced by captopril has an effect on the release of immunoreactive ET-1 (irET-1) from human umbilical vein endothelial cells. The effect of captopril on irET-1 release was compared to that of the AT1/AT2 receptor antagonists losartan and CGP42112. Furthermore the effect of nitric oxide on irET-1 release was investigated. Results: Captopril reduced irET-1 release of cultured endothelial cells in a time and concentration dependent manner (60 ± 8 fmol/10^5 cells vs control 85 ± 6 fmol/10^5 cells after 72 hours, $p < 0.05$). The AT1/AT2 receptor antagonists losartan and CGP42112 reduced irET-1 release in a concentration dependent and additive fashion (losartan 46 ± 2fmol/10^5 cells, CGP42112 47 ± 3 fmol/10^5 cells, losartan and CGP 40 ± 3 fmol/10^5 cells vs control 50 ± 4 fmol/10^5 cells, $p < 0.05$). The reduction of irET-1 release by captopril was not augmented by addition of losartan and CGP42112. The inhibition of nitric oxide synthesis by N^G-monomethyl L-arginine resulted in a minor increase in irET-1 levels. The increase of nitric oxide levels by sydnonimine had no influence on irET-1 release. Thus, it is concluded that captopril reduces endothelin-1 release of cultured endothelial cells predominantly by decreasing angiotensin II synthesis but not by stimulation of the bradykinin/nitric oxide pathway.

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